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Genetic Determinism : How Not to Interpret Behavioral Genetics

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Genetic Determinism

How Not to Interpret Behavioral Genetics

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ABSTRACT. Recently, investigators in behavioral genetics have found loci on the genome (so-called 'quantitative trait loci' or QTLs) that are associated with complex mental traits, such as anxiety or novelty seeking. The interpretation of these findings raises interesting theoretical questions. At first sight, the discovery of 'genes-for-personality' seems to support genetic determinism and reductionism. Genetic determinism is the view that the phenotype is precoded in or determined by the genotype. However, evidence from developmental biology and neural modeling indicates that development is a result of interactive processes at many levels, not only the genome, so that geneticism must be rejected. Identifying QTLs and perhaps also the causal paths in the tangle of top-down and bottom-up influences between genome, organism and environment is best seen as a simplification. It amounts to considerably less than reduction in the classical sense of replacement via bridge laws or elimination. It is argued that higher (psychological and physiological) levels are functionally characterized and are irreducible to molecular-genetic levels. Therefore, it is to be expected that ideas about inter-level relations may be useful in clarifying the relation between loci on the genome (QTLs), gene products, the nervous system, behavior and personality, and to help identify the contribution of genetic factors in behavioral genetics.

KEY WORDS: behavioral genetics, determinism, developmentalism, genetics, reduction

Introduction: Genetic Determinism

It has been known for some time that diseases such as Huntington's disease, phenylketonuria (PKU) and others have genetic origins. More recently, behavioral geneticists have reported that also many cognitive capacities and personality traits have a large genetic component (Plomin & DeFries, 1998; Plomin, DeFries, McLearn, & Rutter, 1997). Some of these results, for example the heritability of IQ, have been a source of bitter disagreement (e.g. Block, 1995), and some, like the presumed discovery of the 'gay gene', made headlines (see Hamer & Copeland, 1998).

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The background of these debates seems to be the dark feeling that biology is destiny, that what was thought to make up human dignity, mood, personality, free will, self-determination, is determined by blind molecular and physiological mechanisms. Sometimes, these results have been interpreted in terms of genetic determinism: genes are a kind of blueprint or program from which our mental and physical make-up develops, so that genes 'pull the strings' of our skills and motives, and behavior is controlled by genes (e.g. Peters, 1997; cf. Lewontin, 1993).

The purpose of this paper is look at some of the assumptions and implications of recent work in behavioral genetics. It will be argued that genetic determinism is a simplistic view and rests on a misconception of reduction, and on a mistaken idea of the role of genes determining the phenotype.[1]

Behavioral Genetics

Behavioral Genetics: Twin Studies and Path Analysis

Traditionally, behavioral genetics has attempted to attribute variance in phenotypic traits in a population to either genetic or environmental sources. Its methods involve analysis of covariances of twin pairs on a number of behavioral indices (intelligence, smoking, neuroticism, schizophrenia, alcoholism, etc.). The basic logic is that monozygotic twins are completely identical genetically, while dizygotic twins share about half their genetic material; assuming that the environmental component is approximately equal for both, higher covariances in monozygotic twins can be interpreted as indicating strong hereditary influence (Plomin et al., 1997).[2]

More sophisticated analysis involves model fitting; starting with a simple model (e.g. all variance is due to unique environment), statistical tests indicate whether including more variables (e.g. common environments and genetic factors) produces significant differences from the simple model, and thus warrants the conclusion that they contribute to the trait. Models are fitted in a hierarchical fashion: usually, one starts with a model which assumes that all variance is due to common environment, then it is tested whether a model including (additive) genetic factors makes a significant difference, and next whether unique environment does so.

Typical findings of twin research are that alcohol use is more hereditary than environmentally determined, and more so at later ages (Koopmans & Boomsma, 1996), or that the influence of the environment on IQ scores decreases with age (Plomin et al., 1997, pp. 146–149).

The contribution of genetic factors is expressed in the so-called 'heritability quotient', the percentage of variance in a trait that can be attributed to genetic factors. The remaining variance is then attributed to common

environment and unique environment. For IQ, it is about 50 percent (somewhat less in children, somewhat more in adults); about 25 percent is due to shared environment, the other 25 percent to unique environment and measurement error (Plomin et al., 1997, pp. 145, 150).

This approach has drawn some fire, partly for political reasons (as much of the *Bell Curve* debate shows—see Jacoby & Glauberman, 1995), partly for conceptual ones. A first problem is that measures like the heritability quotient refer to a population, roughly in the same way as an average or a variance, and cannot legitimately be applied to individuals. (Van der Steen [1999] collected a number of examples of misinterpretations ignoring this basic principle.) However, if the heritability quotient is high, it is reasonable to assume that the proportion of variance accounted for by genetic factors is considerable.

Another pitfall is that heritability coefficients cannot be generalized beyond the sample. For example, in a (hypothetical) completely egalitarian society, where all individuals were subject to exactly the same environmental influences, heritability would be 100 percent (minus noise). Of course, this would prove very little as to the role of heritability in other environments, and it certainly would not prove that genes are the sole causes of behavior (Block, 1995; Van der Steen, 1999).

A particularly vicious mistake in this context is to conclude from a relatively high heritability within two groups (e.g. Caucasians and African-Americans) that the differences between groups (e.g. a reliably higher IQ in whites) are also genetically determined to that extent (Block, 1995). It is important to remember then that no conclusions about the causal path from genotype to phenotype can be drawn from heritability coefficients.

To put yet another misunderstanding aside: the fact that some trait is genetic does not mean that it is not susceptible to environmental interventions. The case of PKU, one of the first where the genetic causes and the remedy of a debilitating disease were discovered, is instructive: this condition is entirely genetically determined, but the symptoms can be prevented by environmental means, namely a strict diet. A more technical problem with heritability quotient, sometimes put forward by developmentalists (see below), is about the additivity assumption. Quantitative genetics tries to separate the relative contributions of environmental and genetic components, assuming that these are additive. For this reason, Wahlsten and Gotlieb (1997) question the validity of the technique of separating nature and nurture (genetic and environmental variance). However, this criticism of behavioral genetics may be exaggerated. It seems generally accepted that at least some part of the phenotypic variance can be attributed to gene–environment interactions. Furthermore, behavioral genetics can accommodate polygenic models where environmental and genetic influences (dominance and epistasis) can be entered as parameters in the models to be fitted, and their contribution to the fit can be tested. Thus, one might argue that whenever

additive effects contribute considerably to phenotypic variance, and when adding dominance and epistasis doesn't produce a significantly better fit, the latter can safely be ignored. Presumably, developmentalists would counter that the fact that an (additive) model yields a good fit does not prove that it captures the underlying dynamics, and that additive models are a fundamentally wrong approach to interactive developing systems. Hence, even finding robust additive environmental and genetic variance components may fail to convince the developmentalist.

The cautious conclusion is that modeling in behavioral genetics, as elsewhere in psychology (cf. Dolan & Molenaar, 1995), is tentative and subject to validation, by (among other things) replication and by producing consistent and interpretable results. Behavioral geneticists' use of additive linear models is such a technique to test hypotheses about contributions from genetic and environmental sources to development.

To sum up then: heritability allows no conclusions about race; nor about individuals and their characteristics; nor about the contribution of genetic and environmental factors beyond the population sample. And no-one knowledgeable in behavioral genetics will pretend otherwise. Cautious interpretation will prevent such rash and unwarranted conclusions about the role of genes in behavior; in that case, there will be no reason to denounce (responsible interpretations of results of) behavioral genetics as ideology (cf. Allen, 1994; Lewontin, 1993). Once the confusion about the nature of heritability quotients is cleared, nasty debates about racism can be put aside. Therefore, political and ideological issues will be deliberately ignored in the rest of this paper. The really interesting theoretical issues in behavioral genetics lie deeper: What is the nature of genetic determination? To what extent are genes the driving force behind the shaping of the phenotype?

Recently, behavioral genetics has moved beyond indirect estimates of heritability; more direct measures of the genotype have become available, and loci on the genome can now be related to personality traits or temperament (for a popular account, see Hamer & Copeland, 1998). Also, educated guesses can now be made about the connection between genes, gene products, such as neurotransmitters, and behavior.

Thus, at first sight, the new (behavioral) genetics may be able to trace the causal path from genotype to phenotype, and thus show how behavior, skill and personality are determined by the genome. Let us have a closer look at the methods and underlying assumptions of this line of research.

The New Behavioral Genetics: QTLs

Until recently, most progress in genetics was made in discrete phenotypic traits associated with a single gene. Huntington, some types of Alzheimer, PKU, Fragile X syndrome, are discrete single-gene traits, where one gene controls the presence or absence of a trait or disease. Quantitative traits, in

contrast, are determined by multiple loci on the genome, and their phenotypic variation is continuous rather than discrete. Psychologically interesting cases of polygenic complex traits are cognitive abilities (intelligence, dyslexia), and temperament and personality. The loci controlling these traits are referred to as QTLs (quantitative trait loci) (e.g. Martin, Boomsma, & Machin, 1997; Tanksley, 1993). After the successes in single-gene traits, genetics is now looking for the more complex cases. Progress in molecular genetics has made it possible to map ever larger portions of the human genome. Thus, more direct measures of the genotype have become available: quantitative genetics uses statistical methods to estimate the relative contribution of loci on the genome to quantitative traits. QTLs then refer to multiple loci that affect complex quantitative traits. Molecular markers, DNA sequences known by acronyms like RFLP, VNTR, SSR (which indicate, for example, how often a particular sequence of base pairs is repeated), can be used to mark a locus on the chromosome, and thus to map the polygenes underlying such traits (Tanksley, 1993).

QTLs can be detected through linkage analysis or through allelic association. Linkage analysis looks for shared DNA markers in family members. Clinical genetics attempts to find the genetic causes of diseases by looking for the co-occurrence of a genetic marker and a trait. Co-segregation of a marker and a disease within families indicates that the gene for that disorder lies close to, or is identical with, that locus. Allelic association studies look for a correlation between an allele and traits across a whole population. Linkage studies have so far mostly been done in single genes and dichotomous traits (Plomin, 1997), though it can in principle also be applied to continuous traits.

Recently, not only single-gene, dichotomous, but also continuous, complex (polygenic) traits—e.g. intelligence or anxiety—have come under scrutiny. Such studies try to find QTLs that account for some proportion of the variance in a continuous quantitative trait. The result is a battery of markers each of which accounts for a proportion of the variance on that trait (Plomin et al., 1997, p. 95). The method consists in finding those regions on the genome that are shared between affected individuals, but not between those unaffected (Lander & Schork, 1995). Such a marker can be a QTL or lie adjacent to it. Since a gene codes for protein synthesis, the next step is to zoom in on the gene itself and study the gene products that are involved in the production of some phenotypic trait.

An Example: Temperament and Personality. An interesting aspect of these developments is thus that gene products can be related to psychological traits. For several personality traits in mice, corresponding loci on the genome have been found (including one for good motherhood). For example, recently discovered QTLs for emotionality in mice thus suggest a bridge from molecular genetics to mind and behavior. Open field activity in mice is

associated with regions of chromosome 1, 12 and 15 (Flint et al., 1995). The mice's genome is supposedly homologous with humans, and therefore these findings could to some degree be generalized to humans (Flint & Corley, 1996), indicating a genetic basis for susceptibility to anxiety or neuroticism (Flint et al., 1995). Lesch et al. (1996) reported that anxiety may be related to a serotonine transporter gene: a more efficient serotonine transporter (better re-uptake) was associated with less anxiety.

Novelty seeking can be related to one of the dopamine receptors on chromosome 11 (Benjamin, Patterson, Greenberg, & Murphy, 1996; Cloninger, Adolfsson, & Surakic, 1996). Ebstein et al. (1996) found that variance in number of repeats in the D4DR dopamine receptor gene is related to novelty seeking. (However, only 4 percent of the genetic variance is accounted for by this genetic polymorphism.)

Hamer and Copeland (1998) sketch a broad view of the influence of genes on temperament and personality, focusing on harm avoidance (including depression, melancholy anxiety) and on novelty seeking (these are two of the four dimensions of temperament, proposed by Cloninger et al.; the other two are reward dependence and persistence). Psychopharmacological research suggests that dopamine is related to novelty seeking. Serotonine is associated with harm avoidance, and is involved in depression, anxiety, anger and hostility. Apparently, its function is to make people feel bad (so that they may do something about it): hence, it could be called a kind of punishment drug (Hamer & Copeland, 1998, p. 103).

It should be borne in mind that these results are still speculative, and await confirmation. Geneticists emphasize that, so far, little more than statistical correlations have been found, and the intermediate steps from genes to gene products to neurophysiology to behavior are still a matter of speculation (e.g. the notorious 'gay gene' is no more than the finding that a specific marker is shared [significantly] more frequently than the expected 50 percent between homosexual brothers in a few dozen cases—Hamer & Copeland, 1998, p. 194). Furthermore, even more importantly, it is usually considered misleading to talk about genes as complete blueprints or programs for finished organisms (Hamer & Copeland, 1998; Kitcher, 1997). The expression 'gene for' should be interpreted as implying a background of cytological, organismic and environmental resources. For example, that fruitflies have a gene for red eyes does not mean that a locus on the genome will produce undetached red eyes all on its own (Sterelny & Kitcher, 1988).

What makes these new approaches in behavioral genetics interesting is that the genotype can be screened directly rather than through the indirect method of twin studies. This means that genetic variation can in principle be related directly to behavioral variation, and it seems that thus on the individual level the inference from chromosome to behavior can be made. For example, in the studies on neuroticism (Lesch et al., 1996), one allele of the gene that encodes for a serotonine transporter results in more protein,

more serotonine uptake and less anxiety and neuroticism, the other allele in less protein and more neuroticism. The results mentioned above may have to be qualified, pending replication, but the principle seems robust enough.

The Problem. At first sight, such findings may give rise to the idea that at least some of the genes that affect behavior have been identified, and therefore that genes determine development, inevitably leading towards certain types of behavior or personality. Some see behind these developments the bogeyman of genetic determinism, or the Gene Myth: the view that our nature (or even our fate) is in the genes, that genes determine behavior like a puppeteer his puppets (Kitcher, 1997, chap. 11; Peters, 1997, pp. 5–7). This raises at least two interrelated theoretical problems. First, these developments seem to suggest that traits are precoded in a kind of blueprint in the genes. Secondly, they raise the question whether higher-level properties, namely behavior and mental capacities, can (in some sense or other) be reduced to genes. Both issues touch upon classical theoretical issues. The second gives a new twist to debates on reduction and elimination in psychology, and relates these to philosophical work on the relation (reducibility) between molecular and classical genetics (Kitcher, 1984/1994; Waters, 1990/1994). The first issue relates to debates in developmental biology, where the Gene Myth has been heavily criticized.

Reduction and Genetic Determination: Three Positions

With respect to reduction and decomposition in behavioral genetics, three positions can be distinguished. The first is the anti-reductionism exemplified in developmental systems theory: development is a tangled web of many resources, where the genome has no pride of place. Its antithesis is reductionism in developmental biology, which holds that the embryo can be 'computed', as a cascade of causal molecular processes. The third view agrees with the developmentalists that there is no traceable causal chain from genetic blueprint to organism, but nevertheless accords some privileged explanatory role to genes.

Developmental Systems: Morphogenesis vs Blueprints

As mentioned, talk about 'genes for'—e.g. for crime, intelligence, homosexuality, etc.—is either a short-hand for a complicated issue (Sterelny & Kitcher, 1988), or just sloppy phrasing. It seems widely accepted that genes do not control development as deterministic specification of phenotypic traits, and that the genome is not something like a blueprint containing instructions specifying the resulting phenotype, including its psychological traits.

Developmentalists like Gottlieb (1991; Wahlsten & Gottlieb, 1997) and Oyama (Van der Weele, 1999) are more radical in their rejection of genetic explanations. They stress the interactionist nature of development where genes and environment are inseparable: information is not coded in genes; rather it emerges from gene–environment interactions, and what information genes carry depends on the timing and context of development.

Wahlsten and Gottlieb (1997) argue that the attempt at separating nature and nurture in behavioral genetics ignores the essential interdependence of genes and environment. Extrapolating from animal research to human development, they purport to show that genes do not act on their own, and that development is not completely specified in advance in genes and environment. More precisely, they argue that development results from interactions between levels: genes, cells, the organism and its social environment. The developmental systems approach they advocate focuses on the dynamics of developments as it results from interactions between levels of environmental (physical and social), behavioral, neural and genetic activity. Explaining development is a 'multilevel affair involving at least culture, society, immediate social and physical environments, anatomy, physiology, hormones, cytoplasm, and genes' (Wahlsten & Gottlieb, 1997, p. 183).

This approach involves a hierarchical and co-actional model of these levels. Causality is considered as bidirectional, moving both ways between a hierarchy of levels: gene expression is influenced by the cell environment, and the cell by the external environment, which is influenced by the organism's behavior, and all the way back. Not only is the phenotype determined bottom-up from genes, the meaning of the genetic information is also determined top-down from the environment and the whole organism. Finally, development is not deterministic (Gottlieb, 1991); a norm of reaction, picturing different outcomes for different genotypes in different environments, is the preferred way of analysing development.

Developmentalism is anti-reductionistic, as, for example, Goodwin (1994) makes explicit. In his view, genic reductionism amounts to the disappearance of the organism. The genocentric view that genetic programs code for the molecular composition of organisms considers organisms as synthesized from molecules built by a genetic code; it means that organisms are de facto reduced to mere vehicles for survival (ultimately, the survival of genes). Goodwin suggests that this is just a metaphor, which might, and should, be replaced by a better paradigm, focusing on emergent organisms with their own intrinsic value. Such an alternative allows a different perspective on developmental biology. It is based on the idea of morphogenesis, the process whereby new properties emerge at the organismic level. The morphogenetic view implies that organisms are self-organizing forms. Thus, evolution is not only driven by natural selection, the essentially historical and contingent adventures of genes, but is also subject to general principles of biological form.

Genes are no blueprint for development, since they have no effect outside the organized context ('the generative field') in which they act, and cannot be separated from the total pattern unfolding in space and time. It is not even true that the phenotype develops from the genotype; both coexist in time, and influence each other continuously. According to Goodwin (1994, pp. 133, 139), genes define the region of parameter space where development (considered as trajectory through that space) starts. Thus, the way genes act is by selecting patterns; they cooperate with generic forms to produce variations in form, rather than controlling development.

This leads to a different view of organisms: 'the irreducible entities that are engaged in the process of generating forms and transforming them by means of their particular qualities of action and agency, or their causal powers' (Goodwin, 1994, p. 176). Autonomy and agency is an emergent property of organisms that is irreducible to molecules; self-reproduction is not in the genes, but is a property of a whole integrated organism. An organism can be credited with agency and causal powers in its own right, not just as derivatives of genes, and with a particular nature that allows it to function in its environment (Goodwin, 1994, p. 177). Goodwin thus hopes to establish an independent status for the organism.

To sum up: a different view of organisms as autonomous generators of form, endowed with agency and causal powers, self-expression, can be seen to emerge here, in contrast with the genocentric view, where organisms are both computed from genetic programs, and mere vehicles for the agents of evolution, the selfish genes. The emphasis is the legitimacy of higher levels (Goodwin, 1994, p. 181), and against reduction to a basic molecular level.

The claim Goodwin and others reject is that we can 'compute' an organism (Goodwin, 1994, p. 8), that is, completely reconstruct the causal path from molecule to organism as the result of an algorithmic process of protein synthesis.

Computing the Embryo?

A completely opposite position in the interpretation of the role of genes is Rosenberg's (1997) analysis. He argues that reductionism is standard practice in developmental biology, and that higher-level, emergent, functional considerations play no role in the explanation of an embryo's development.

It seems to have been accepted wisdom for some time in the philosophy of biology that higher-level characterizations like Mendelian genetics have some explanatory power even in view of the progress of lower-level characterizations like molecular genetics (Kitcher, 1984/1994). Complete reduction to lower-level entities was not envisaged.

Whereas in previous work (Rosenberg, 1985, chap. 3), he at least acknowledged the heuristic value of functional analysis, Rosenberg (1997)

now argues that there is (in developmental biology) no such thing as an autonomous and irreducible level of functional explanatory generalizations, let alone that the functional level has any kind of explanatory primacy. In his reconstruction, developmental biology is purely reductionistic: it attempts to track the development from macromolecules to organisms, in terms of an algorithm specifying elements and composition rules. Molecular interactions, presumably a manageable number, acting in cascade, give rise to the macro properties of the embryo, producing cell membranes from nucleic acids from molecules. No more is needed to explain development. Functional and teleological characterizations of development are just manners of speaking that do not explain anything; rather, they have to be explained in terms of gene action. The apparent teleology of the process is the explanandum, not the explanation: functional characteristics are to be discharged by specifying the way such properties are instantiated in the algorithms governing the synthesis of organisms from molecules. Crucial for Rosenberg's denying explanatory value to functional characterization is that developmental biology is hardly interested in general laws and more in tracing singular causal paths. (The idea that causality depends on explanatory interests is rejected.) Within such a causal framework, autonomy for higher levels, and the claim that these might provide better explanations than the lower-level mechanisms, implies that functions would have causal powers. Thus interpreted, functionalism implies top-down causation, which must seem an ontological horror to Rosenberg.

To summarize: developmentalists see no special role for genetic information; rather, they focus on the whole dynamic emergent process. Reduction of development to causally effective blueprints is out of the question. In stark contrast, the algorithmic approach assumes that the organism can be computed, and the functional idiom has no place in explanation.

A Third Option

In a recent series of papers, Clark (1998a, 1998b; Wheeler & Clark, 1999) has called attention to the parallelism between representations as explanation of behavior, on the one hand, and genes as explanation of phenotypical development, on the other. In the philosophy of mind and cognition, dynamicists (e.g. Van Gelder, 1998) have questioned the orthodox view that internal representations code for the goal to be achieved and the behavior to achieve it, and can be cited as the causes of intelligent, goal-directed behavior. Recent work in robotics and animal behavior suggests that a complete central representation of behavior is just impossible (cf. Keijzer, in press; Looren de Jong & Sanders, 1990). Some theorists (e.g. Brooks, 1995) have concluded that the concept of representation or neural program is useless as an explanation of behavior. The role of genes in explaining the phenotype shows some remarkable conceptual similarities. The interactionist

position emphasizes that the causal complexity of the web of causes and interactions makes it impossible to isolate processes that code for phenotype, or behavior, respectively. Clark calls this view, both in cognitive psychology and in developmental biology, egalitarism: all causes are considered equally important as explanations, or, even more radically, the focus is on interactive patterns rather than individual causes. The point is that there is no pre-existing specification of behavior or phenotype, but that both result from a tangled web of interactions, in which coupled organism and environment, genes and their context mutually codetermine each other. In this multitude of interacting forces, the gene is more like a catalyst, a conductor rather than the whole orchestra (Elman et al., 1996), or the parameter in a dynamic system rather than the driving cause. Unlike programs, which are supposedly self-contained repositories of information, genes are part of an environment of processes, without which they do or mean nothing.

This implies that notions of representation as specification of behavior leading to a final goal state cannot serve as explanations. The same goes for genes as programs or specifications of a phenotype. Causal complexity and context dependency not only preclude meaningful talk about 'genes for'; what makes decomposition (see Bechtel & Richardson, 1993) in semantically interpretable units (such as units for reading and copying DNA, or routines for evaluation of goals) problematic is what Clark calls Continuous Reciprocal Causation: mutually co-determining interaction between all components.

However, as Clark points out, egalitarism with respect to causes does not follow from the fact of causal complexity. Complexity and reciprocal causation is in itself no reason for skepticism about the existence of genes or representations. A representation or code does not have to be self-contained nor does explanatory relevance have to be measured by the causal contribution it makes. That a blueprint or code is not the only or most important factor in the causal web does not mean it could not be legitimately singled out in an explanation. Clark calls this the primary locus of plasticity: the change of a factor would make a difference on the phenotypic outcome, against an ecological backdrop of other factors. A gene represents an outcome because it has the (selected) function of bringing this effect about, not because it contains explicitly all the relevant information. Thus, causal explanations may refer to the influence of some factor while 'piggybacking' on the background events in the causal flow.

This, however, leaves the problem of 'causal (and explanatory) spread' (Wheeler & Clark, 1999): when an organism exploits the environment, and its behavior becomes a complex outcome of interactions between body, brain and environment, complex interactions become intractable, and causes spread across systemic boundaries. An example is infant walking, which is apparently a self-organizing system, exploiting by propriocepsis of stretch in the limbs, not directed by a neural motor pattern. Forces in the environment

and the mass-spring system in the limbs are equally causal, and none is explanatorily prior. If we admit that genes do not code for traits, but only set parameters for the developmental process, then a multitude of influences could equally be called parameter setters, and the genetic code has no privileged role as encoding, information or specification that might set it apart from merely causal factors like single-neuron or lowly muscle stretch receptors. Obviously, causal codetermination doesn't suffice for (cognitive or genic) representation.

Wheeler and Clark (1999, p. 128) formulate three criteria for genuine representations, as distinct from mere codetermination. Representations are states with the functional role to bear content, and this role is characterized, first, by arbitrariness: the genetic code carries content in a way that could have been different with respect to its material realization (e.g. genes could have been coded in silicone as long as the relation between triplets and amino acid has the same mapping). Second, there is a 'consumer' of this information, a process that uses it (in the case of genes, the processes of transcription [DNA to mRNA] and translation [RNA to amino acids]). That is, genes can be said to have the function of preserving and transferring information, of coding and decoding, not just brute force. Third, (genic) representations should have some systematic combinatorial structure. Thus, functional role is the characteristic that sets (genic) representations apart from mere causal codetermination. Below, we will return to the issue of functional explanation in the philosophy of science.

Conclusion

In sum, the developmentalists may be right that complexity and multiplicity abound, but that does not preclude an explanatory role for genes. Talk about 'genes for', then, is a way of looking at a tangled web, and singling out the explanatorily relevant strand that has the function to carry information. For the philosophy of mind, Clark (1998b) proposes a kind of physical/informational double-aspect theory: the informational aspect can have different bearers, including dynamic systems and their temporal trajectories. This idea is easily transposed to the issue of genetic determination and the role of genes. My suggestion, to be elaborated more fully in the final section, is that a functional point of view, interpreting what a lower-level mechanism does in the context of an organism and its environment, will flesh out the informational aspect. Thus, we can legitimately interpret a sequence of base pairs as a code for a phenotypic trait, or a neural pattern as representation of an event in the environment. Also, it is entirely legitimate to interpret causal chains of molecular reactions in functional idiom as executing some subtask, like reading or copying DNA.

In this way, we have sketched a middle course between developmentalism and algorithmic reductionism. Against reductionism, we think that to see

what an algorithm for protein synthesis does, we need a higher-level perspective, and against developmentalism, we think that some causal strands are indeed privileged as explanations, namely those picking out causes that make a (functional) difference.

QTLs are examples of complex traits resulting from a tangle of factors. Hence, the framework sketched above should shed some light on the nature of genetic explanations in psychology. Let us look at two empirical approaches relevant to the issue of reductionism vs developmentalism. Elman et al. (1996) tried to flesh out developmentalist claims using neural networks. Schaffner (1998) has refuted radical developmentalism in the case of the behavioral genetics of *Caenorhabditis elegans*.

Two Empirical Approaches to Developmentalism: Neural Networks and *C. elegans*

Neural Networks and Developmentalism

The developmentalist position is supported by Elman et al.'s (1996) neural modeling. They agree with the developmentalist rejection of genetic preformationism: genes are no static blueprint precoding for phenotype. The way genetic information specifies some properties of behavior and knowledge is, in the view of Elman et al., by a plethora of interactive processes. The phenotype is the result of interaction between genetic constraints and the context: experience, the environment, other genes and the whole organism.

Elman et al. make two important points. The first is that even apparently innate behavior may result from interactions between genes and environment, rather than being precoded in the genes. For example, the specialization of cortical regions for specific tasks (e.g. ocular dominance columns), although almost invariant across individuals, is a product of postnatal environmental tuning. Second, interactions take place within and between different levels. More precisely, interactions occur between cells in brain development, between the developing brain and environmental stimuli as in maturation, and between the brain and cultural influences.

Elman et al. corroborate these claims by providing neural network simulations of development. They present a model of chick imprinting and recognition at the level of brain systems, using neurophysiologically plausible mechanisms like lateral inhibition and recurrent activation to produce recognition of spatio-temporally continuous objects.

The conclusion Elman et al. draw from these simulations is that their neural network has no prespecified representations; the knowledge it acquires is not innate in the sense of precoded, but results from architectural bias in interaction with environmental input. In these simulations, context

and timing are crucial. Differences in the degree of plasticity of cortical regions (maturational sensitivity) give rise to functional architectures as an emergent result of input and differential maturation rates. A gene is not a self-contained instruction set; rather, what information genes contain crucially depends on the interactive process they are part of.

Elman et al., like Clark, assume an analogy between mental representations and genetic codes, and claim that their neural network simulations of development throw light on the possibility of emerging order without preceding. For the interpretation of behavioral genetics the conclusion from these simulations is that no simple complete and reductive explanation for behavior from genetics seems possible. The evidence from neural modeling indicates that the idea of genetic determinism is empirically unsound. However, as argued above, that does not justify wholesale rejection of genetical explanations. It might be worthwhile to look for vehicles of codes and representation in the dynamic temporal patterns that make up the physical working of the network (Clark, 1998a, 1998b).

That leaves the question of how to interpret the relation between QTLs, gene products and behavior if neither reduction nor all-out developmentalism is an option.

C. elegans

In a recent paper, Schaffner (1998) provides a careful analysis of developmentalist claims. Reviewing the experimental evidence on *Caenorhabditis elegans* (a small worm with a simple and thoroughly studied nervous system), he concludes that in this organism many genes affect one neuron, and one gene can influence several neurons (pleiotropy); that different environments cause differences in gene expression, which in turn cause different behaviors in presumably genetically identical organisms. (Incidentally, *C. elegans* is the first organism whose genome has been completely mapped; also, its complete anatomy is known. What is not known is how these genes cooperate to build the nematode.) Briefly, Schaffner's conclusion is that pleiotropy, plasticity and environmental effects abound and these preclude simple gene-to-neuron-to-behavior mappings. For *C. elegans*, developmentalists are correct in emphasizing the prevalence of context effects, and in rejecting preformationism. However, all-out developmentalism that considers genes and environment as a single inextricable unit must also be rejected, according to Schaffner: genes are special in the sense that they have 'informational priority', and their causal role can be separated out. Also, the experimental evidence gives no reason to adopt nondeterminism. So, in the very simple case of *C. elegans*, genetic determinism and preformationism must be rejected. It seems reasonable to extend this to human behavioral genetics. The conclusion that can be drawn from this brief discussion of developmentalism in biology is that there is no simple causal

path from genotype to phenotype, and that the latter is not explained by or reducible to the latter. Rather we see a tangle of causal influences within and between levels. The error of the Gene Myth is that it tries to jump from the molecular to the psychological level, ignoring the interactions between levels.

Reduction and Function

Reduction and its Discontents

Recent work in the philosophy of science has almost unanimously rejected the classical view of reduction, based on the hackneyed example that temperature is (reducible to) average kinetic energy (Churchland & Churchland, 1994; Hardcastle, 1992; McCauley, 1996). Rather, a number of alternatives have been proposed that focus on inter-level relations.

Churchland and Churchland (1994) argue that the dichotomy of classical reduction and elimination is a simplification of the possibilities of inter-theoretic relations. Between 'smooth' reduction, where the older theory is entirely preserved in the reducing theory, and elimination, where it is entirely abandoned, lies a continuum of more or less 'bumpy' reductions, where the reduced theory is corrected to some degree, and the referents of theoretical concepts may be fixed in different ways (see Schouten & Looren de Jong, 1998). Wimsatt (1984) and McCauley (1996) distinguish these inter-level reductions between successive theories from intra-level reductions where mapping between phenomena at different levels is sought—for example, one might try to map patterns of Mendelian inheritance onto molecular phenomena. Failure of such mapping amounts to irreplaceability of the upper level (whereas in intra-level contexts incommensurability is a reason for abolishing the old theory).

Wimsatt (1976) makes a forceful case for distinguishing irreducible levels in reality, characterized by local maxima of predictability and order, and he points out that belonging to a level is a real property of objects. Whereas higher levels can ideally be characterized as being composed of lower-level entities, in biology and psychology categories typically cross-cut levels, and no nice hierarchy, or neat mapping relations between higher- and lower-level phenomena, are to be expected, although illuminating local connections can sometimes be made. Thus, some phenomena simply have no explanation at lower levels—for example, Mendelian inheritance does not.

Furthermore, Wimsatt (1976, 1984) was one of the first to introduce pragmatic considerations into the analysis of reduction. Explanation is context-relative: it answers 'why' questions against a background of assumptions and unasked questions; relevant factors are singled out from a lot of other possible but currently uninteresting or ignored causes. In the

classical D–N account of reduction, all sorts of things can be deduced that are irrelevant as explanatory factors (Hardcastle, 1992).

Kitcher (1984/1994) shows that classical Mendelian genetics cannot be said to be reduced to molecular genetics. Rather, some of the problem-solving capacities of one theory can be generated from another theory. This notion of inter-level relations, which Kitcher calls explanatory extension, is much weaker than classical reduction. In contrast with the classical notion of global theory reduction, it may hold between no more than fragments of theories at different levels, as in this case between molecular and classical genetics. The extending theory may show how problematic assumptions of the extended theory are possible, or it can conceptually refine the extended theory by changing what theoretical predicates concepts refer to (Hardcastle, 1992, p. 419). Interestingly, the extending theory may also be the higher-level theory: functional psychology influencing neurophysiological theorizing is a case in point (Schouten & Looren de Jong, 1999). The relations between levels may be described as 'illuminating' each other (Hardcastle, 1992), or as exerting selection pressure on theories across levels (McCauley, 1996). In all these cases, no more than partial and fragmentary reductions or inter-level relations are to be expected (but cf. Waters, 1990/1994). Such ideas render implausible the reductionist intuition behind genetic determinism.

Levels and Functions

An alternative may be found in a multi-level perspective, where the higher level is functionally characterized; the functional account addresses what a process does (or should do, or was selected to do) in the context of an organism and its environment (Bechtel, 1986; Looren de Jong, 1997). If such a perspective has any plausibility, the algorithmic account of developmental biology must be missing something. This can be shown using Gasper's (1992) notion of multiple supervenience. This is the opposite of multiple realizability (a function can be realized in different physical substrates): it holds that the same physical substrate can support a number of different higher-level properties. For example, the same molecular base (the 'supervenience base') in a piece of metal supports a number of different dispositions such as electrical conduction, rigidity and opacity. Which of these counts in a given context (whether a piece of aluminum functions as a ladder because of its rigidity, or whether it will function to electrocute its user when touching a power line because of its electrical conductivity—the example is from Jackson & Pettit, 1990, p. 204) cannot be seen from the lower-level perspective. Hence, not all information is specified in the lower-level description, and most of the supervenient dispositions are not explanatorily and causally relevant. To see the 'real pattern' (Dennett, 1990), a higher-level perspective is required that picks out the (causally) relevant

aspects. Multiple supervenience suggests that the component's function, that is, what it does (or is supposed to do), is not visible in the lower-level structural description. Functional descriptions indicate which aspects of lower-level descriptions will be singled out as relevant for explanations. Information salient at the macrolevel is 'objectively lost' (Gasper, 1992, p. 668) when we descend to lower levels.

In Gasper's example, cytological explanations highlight connections which are invisible at the molecular level:

> The central connections in the cytological explanation will be relations of causal relevance. The explanation will tell us what it is about—for example, a chromosome which leads it to give rise to certain effects in a particular set of circumstances. The chromosomal characteristics in question will supervene on a particular molecular base, but citing this base will not adequately capture the relevant characteristics since these will not be the only properties that supervene on it. (Gasper, 1992, pp. 668–669)

So, a function is more than the causal transactions in which it is realized. It also includes relational and contextual factors. Without knowing what the lower-level mechanisms are supposed to do, that is, without a higher-level perspective, we would be missing what it has been selected for, what it contributes to the functioning of the system as a whole, and what its significance is in the context of an organism and its environment. So, *contra* Rosenberg (1997), higher-level functional characterizations are in a sense objectively real, and functions are explanatorily relevant.

Interpreting QTLs: Function vs Reduction

Findings about QTLs, loci on the genome that influence complex mental traits (loosely: 'genes for'), are best interpreted as a simplification (Schaffner, 1998) that yields interesting predictions about phenotypic differences on the basis of molecular genetic differences. Such a simplification, or selection from the supervenience base, of molecular-genetic mechanisms is guided by considerations of what these mechanisms do in terms of gene products relevant for physiological processes, neurotransmitters, temperament, behavior, and so on. This is not classical reduction since it focuses on only one causal strand, the genetical, ignoring others. The genes are not sufficient in themselves to exhaustively explain or reduce the phenotype; since the path from genotype to phenotype is not a complete causal sequence, it will not yield a complete replacement (via bridge-laws) or an elimination of the latter by the former.

Schaffner (1998) emphasizes that such simplifications in behavior genetics, identifying single causal patterns in the tangle of developing systems, are to be highly valued. One strategy in explaining complex systems is Bechtel and Richardson's (1993) decomposition and localization. The decomposition strategy looks for sub-processes, small in number and with

minimal interaction; localization tries to identify functions as found by decomposition with system components.

The functional approach sketched above seems an appropriate framework for the new behavioral genetics. As we have seen, the lower-level causal paths, tangled and complex as they are, can be interpreted in terms of their contribution to explanations of the physiological and psychological properties under consideration.

In fact, actual research into QTLs focuses on co-occurrence of behavioral and molecular-genetic properties and tries to assess the degree of shared variance, ignoring a host of other loci on the genome (the rest of the supervenience base). In this sense it involves a double-aspect endeavor (Clark, 1998b), looking for the lower-level (physical) realization of higher-level functional properties.

In conclusion, on the one hand, we have found no support for the wholesale (classical) reduction genetic determinism demands. Genetic determinism seems to rest on an obsolete model of reduction and explanation. Identifying QTLs can best be interpreted in terms of inter-level relations like decomposition and localization or explanatory extension, where illuminating relations between different domains are established, in this case between molecular genetics and psychology. On the other hand, the developmentalist rejection of privileged explanations ignores that interesting generalizations can be made by simplifying and selecting parts of the supervenience base. The psychological or physiological provides the functional perspective, showing where to look for psychologically 'real patterns' in molecular and neurophysiological mechanisms.

Conclusion: Levels, Reduction and Genetic Determinism

We have looked at two major and related problems in the interpretation of the new behavioral genetics, namely genetic determination and genetic reduction. These issues have become more urgent now (behavioral) genetics has begun to identify loci on the genome (QTLs) that are related to personality and mental capacities. This seems to fuel the suspicion that we are our genes, and that genes code for personality and mental capacities. It was argued that such simplistic genetic determinism is misguided. First, the phenotype is not precoded in the genes; rather, development is a dynamic, interactive process, involving all sorts of top-down and bottom-up causal influences between genes, the whole organism and the environment. Identification of QTLs is not the same as finding the causal sequence from gene to behavior. Rather, it is localizing one component (causal path) in a complex system; it is more like simplification than like the reduction of phenotypic

traits to genes. Therefore, second, finding genetic correlates of phenotypic traits is not reduction in the classic sense, but it seems better accounted for by some of the ideas in the philosophy of science mentioned above, that is, as explanatory extension (Hardcastle, 1992; Kitcher, 1984/1994), and decomposition and localization of isolable components and sub-processes (Bechtel & Richardson, 1993).

Such ideas seem to provide conceptual tools to clarify the role of genetic explanations in behavioral genetics. To summarize the ideas sketched in the previous section: in contrast with the classical D–N model, the pragmatic (Hardcastle, 1992) or functional (Wimsatt, 1984) view has divorced reduction from explanation. The pragmatics of explanation suggests that what counts as a cause or an explanation is determined by context. Calling genes the cause of a phenotype is a (legitimate) simplification, abstracting from a background of many interacting causes (Schaffner, 1998). As argued above, the functional level of description is irreplaceable in biology and psychology (Gasper, 1992; Hardcastle, 1992; Looren de Jong, 1997; Wimsatt, 1976); it singles out the upper-level pattern. This means that psychological categories, functionally defined, will not be reformulated or replaced by neurophysiological or genetic ones, but theories at different levels can be seen as illuminating or extending each other. The notion of the co-evolution of theories at different levels (McCauley, 1996; Wimsatt, 1984), or 'partial reductionism' (Hardcastle, 1992), better captures the dialectic of theory development than does reduction.

Applying these ideas to behavioral genetics, we see that behavioral genetics characteristically comprises two levels of description: molecular genetics and behavior. Both have their own problems and explanations, and progress consists not in replacing one by the other, but in establishing bridges between them ('explanatory extensions'). The higher-level perspective allows researchers to group phenomena according to functional criteria that show patterns not visible from a lower-level perspective. Furthermore, since lower-level dispositions (molecular properties) underlie potentially a multiplicity of higher-level phenomena, we need a higher-level perspective to see what dispositions are explanatorily relevant (Gasper, 1992). Thus, in contrast to Rosenberg's reductionism, it is entirely legitimate to constrain the choice of molecular-genetic causal mechanism to those relevant for physiology or psychology; in that sense, genetic causes are interest-relative. The functional or teleological interpretation of loci as contributing the phenotypic, psychological or physiological, level is a case of bridge-building.

Inter-level relations are simultaneously bottom-up and top-down (McCauley, 1996). Not only can higher-level considerations constrain what counts as cause in lower levels, also lower-level discoveries may correct higher-level theories ('coevolution'). Psychological constructs define what

to look for in the genome, and discovery of genes and gene products (neurotransmitter receptors) may suggest revising the psychological theory.

It is to be expected, then, that ideas about inter-level relations (McCauley, 1996) will be useful in clarifying the relation between loci on the genome, gene products, the nervous system, behavior and personality, and help identify the contribution of genetic factors in behavioral genetics. A realistic case of coevolution involving temperament, neurotransmitter receptors and genes might be the search for genetic determinants of personality or temperament (Cloninger et al., 1996) without lapsing into reductionism and geneticism. Future research into the way genetic factors determine psychological characteristics will involve coevolution of genetic, neurophysiological and psychological levels of description.

Notes

1. One school in philosophical psychology maintains that causal explanations of human behavior should be carefully distinguished from explanations in terms of reasons, and that the latter cannot be adequately captured in causal terms. Some authors (e.g. Taylor, 1964; Winch, 1958) emphasize that the vocabulary of action, purpose and meaning is essential to understanding behavior, and is irretrievably lost in a mechanistic account emphasizing physiology, physical movement, causes and laws. Davidson (1963, p. 658) has argued that reasons can be reconstructed as causes, and that explanations that invoke reasons for actions are a species of causal explanation. Whether this position leaves much room for genuine mental causation, distinct from physical or physiological causation, is a much debated point (e.g. Kim, 1998, ch. 2). Obviously, behavioral genetics, and biological psychology in general, are instances of a quest for causes. Some may feel that this amounts to neglecting the real phenomena of human action. However, interesting explanations in the causal domain will be valuable in their own right, even if it is granted that they may be incomplete. In the present author's opinion, it is ultimately an empirical matter whether biological or genetic explanations can (to some extent) explain purposeful rational behaviour.
2. A potential problem mentioned by one reviewer for this journal is gene–environment covariance: certain genes may for various reasons end up more frequently in certain environments, for example smart parents will endow their offspring with smart genes, as well as providing them with an intellectually stimulating environment. This would presumably lead to overestimation of heritability. However, in twin studies individuals with partly (fraternal twins) and completely equal genetic make-up (identical twins) are compared, and environmental and genetic factors can be estimated separately. The environment will then turn up in the model as source of variance, in addition to genetic influences. Also, interaction can (in principle) be estimated. In fact, it can be shown that gene–environment covariance, if not explicitly entered into the model, may behave as an environmental factor, and thus *decrease* the heritability coefficient (Dorret Boomsma & Leo Beem, personal communication).

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